Amendments to the Claims/ Listing of Claims

This listing of claims will replace all prior versions, and listings, of claims in the application.

1. (Original) A method for altering the load of a Hepatitis virus in a host organism infected with said virus, comprising the modulation of the complex formation of a heterogeneous nuclear ribonucleoprotein (hnRNP) K or a functional fragment thereof with a regulatory region on the Hepatitis virus genome.

2. (Previously presented) The method of claim 1, wherein the said virus is selected from the group consisting of mouse Hepatitis virus, woodchuck Hepatitis virus, ground squirrel Hepatitis virus, arctic ground squirrel Hepatitis B virus, human Hepatitis B virus (HBV), duck Hepatitis B virus, heron Hepatitis B virus, sheld goose Hepatitis B virus, snow goose Hepatitis B virus, Ross' goose Hepatitis B virus, stork Hepatitis B virus, woolly monkey Hepatitis B virus, orangutan Hepadnavirus, GB virus B, and human Hepatitis C virus (HCV).

3. (Previously presented) The method of claim 1, wherein the host organism is a microorganism or a mammal.

4. (Previously presented) The method of claim 1, wherein the mammal is selected from the group consisting of a rat, a mouse, a squirrel, a hamster, a woodchuck, an orang-utan, a woolly monkey, a chimpanzee, a tamarin (*saguinus oedipus*), a marmoset and a human.

5. (Previously presented) The method of claim 1, wherein the modulation of said complex formation is achieved by means of altering the total amount of a variant of heterogeneous nuclear ribonucleoprotein (hnRNP) K or a functional fragment thereof in the cell.

6. (Previously presented) The method of claim 1, comprising administering a compound that modulates the complex formation of a hnRNP K protein or a functional fragment thereof with the regulatory region on the Hepatitis virus genome.

7. (Previously presented) The method of claim 1, wherein the regulatory region is enhancer II of a hepadnavirus.

8. (Original) The method of claim 7, wherein the enhancer II region comprises positions 1554 to 1645 of the Hepatitis B virus genome.

9. (Previously presented) The method of claim 1, wherein the said virus is the human Hepatitis B virus.

10. (Previously presented) The method according to claim 1, where the method is an in-vivo method for the identification of suitable compounds that modulate said complex formation.

11. (Original) The method of claim 10, comprising administering a suitable compound for modulating the complex formation of a hnRNP K or a functional fragment thereof protein with the regulatory region on the Hepatitis virus genome.

12. (Original) The method of claim 11, further comprising measuring the number of Hepatitis virus particles in the host organism over a period of time.

13. (Original) The method of claim 11 or claim 12, further comprising:
comparing the obtained results with those of a control measurement.

14. (Original) The method of claim 13, wherein the control measurement comprises the use of a compound that does not modulate the complex formation of said hnRNP K protein or a functional fragment thereof with the regulatory region on the Hepatitis virus genome.

15. (Original) The method of claim 13, wherein the Hepatitis virus is the human Hepatitis B virus, the regulatory region is enhancer II, and wherein the control measurement comprises the use of a variant of HBV that does not contain adenine at position 1752 of the virus sequence.

16. (Previously presented) The method of claim 1, wherein the host organism is a recombinant microorganism expressing a hnRNP K protein or a functional fragment thereof.

17. (Original) The method of claim 15, wherein the microorganism is a cell derived from liver tissue.

18. (Original) The method of claim 17, wherein the cell is of or derived from a hepatocellular or a hepatoblastoma cell line.

19. (Previously presented) The method of claim 18, wherein the cell line is selected from the group consisting of HepG2, Hep3B, HCCM, PLC/PRF/5, Sk-Hep-1, Snu182, HuH-6 and HuH-7.

20. (Currently amended) **[[A]] <u>The</u>** method of claim 1, wherein the complex formation of the hnRNP K protein or a functional fragment thereof with the said regulatory region of the Hepatitis virus is reduced by means of a nucleic acid molecule.

21. (Original) The method of claim 20, wherein the nucleic acid molecule is RNA or DNA.

22. (Previously presented) The method of claim 21, wherein the nucleic acid molecule is selected from the group consisting of an aptamer, a micro RNA (miRNA) molecule and a small interfering RNA (si-RNA) molecule.

23. (Previously presented) The method of claim 22, wherein the nucleic acid molecule is a si-RNA molecule comprising a sequence selected from the group consisting of SEQ ID NO: 2, SEQ ID NO: 4, SEQ ID NO: 6, SEQ ID NO: 8 and SEQ ID NO: 10.

24. (Currently amended) **[[A]] <u>The</u>** method of claim 1, wherein the interaction of a hnRNP K protein or a functional fragment thereof with a regulatory region of the Hepatitis virus is modulated by a compound that modulates the phosphorylation status of cellular components.

25. (Original) The method of claim 24, wherein the compound alters the degree of phosphorylation of a hnRNP K protein or a functional fragment thereof.

26. (Original) The method of claim 24, wherein the compound alters the intracellular quantity of hnRNP K proteins or functional fragments thereof.

27. (Previously presented) The method of claim 24, wherein the compound is an agonist or antagonist for a molecule on the cell surface.

28. (Original) The method of claim 27, wherein the molecule on the cell surface is a receptor.

29. (Previously presented) The method of claim 28, wherein the receptor is selected from the group consisting of a receptor tyrosine kinase, a membrane receptor with associated tyrosine kinase activity, and a G protein coupled receptor.

30. (Previously presented) The method of claim 29, wherein the receptor is selected from the group consisting of a receptor for a platelet derived growth factor, a receptor for erythropoietin, a receptor for tumor necrosis factor, a receptor for leukaemia inhibitory factor, a receptor for an interferon, a receptor for insulin, a receptor for an insulin-like growth factor, a receptor for an interleukin, a receptor for a fibroblast growth factor, a receptor for a granulocyte-macrophage colony stimulating factor, a receptor for a transforming growth factor, and a receptor for an epidermal growth-factor.

31. (Previously presented) The method of claim 27, wherein the agonist or antagonist is a protein.

32. (Currently amended) The method of claim 31, wherein the protein selected from the group consisting of a mutein based on a polypeptide of the lipocalin family binding to a receptor tyrosine kinase, a glubody binding to a receptor tyrosine kinase, an immunoglobulin binding to a receptor tyrosine kinase, a protein based on the ankyrin

scaffold binding to a receptor tyrosine kinase, and **a protein based on the** crystalline scaffold binding to a receptor tyrosine kinase.

33-44. (Cancelled)

45. (Previously presented) A method for treating a Hepatitis infection comprising administering to a subject a compound selected from the group consisting of aptamers, micro RNA molecules, small interfering RNA molecules, compounds that modulate the absolute quantity of hnRNK proteins in a cell, compounds that modulate the degree of phosphorylation of hnRNP K proteins, agonists for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase and antagonists for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase, wherein the viral load is altered via the modulation of the complex formation of a hnRNP K protein with a regulatory region on the Hepatitis virus genome.

46. (Previously presented) The method of claim 45, wherein the agonist or antagonist for a cell surface receptor that is able to induce the regulation of a cellular kinase or phosphatase is selected from the group consisting of a mutein based on a polypeptide of the lipocalin family binding to a receptor tyrosine kinase, a glubody binding to a receptor tyrosine kinase, an immunoglobulin binding to a receptor tyrosine kinase, a protein based on the ankyrin scaffold binding to a receptor tyrosine kinase, a protein based on the crystalline scaffold binding to a receptor tyrosine kinase, a membrane receptor with associated tyrosine kinase activity, and a G protein coupled receptor.

47. (Previously presented) A method for treating a Hepatitis infection comprising administering to a subject a compound identified by a method of claim 8, wherein the viral load is altered via the modulation of the complex formation of a hnRNP K protein with a regulatory region on the Hepatitis virus genome.

48. (Previously presented) The method of claim 45, wherein the Hepatitis infection is caused by HBV.

49-52. (Cancelled).